Materials and Methods: From 8/2005 to 12/2007, 15 patients (pts), 25-61 years old, with high risk acute leukemias, underwent double cord blood transplantation. HHV-6 reactivation was noticed in 7 (47%) at a median time of 40 days (range 25-127) post-transplant. Myeloablative conditioning regimen was used in 5/7. Prophylactic antiviral therapy consisted of acyclovir (n=5/7) or foscarnet (n=2/7). Among them 2 pts had already CMV-infection and 6 (85.7%) suffered from aGVHD and received methylprednisolone at the time of HHV-6 reactivation. Levels of HHV-6 DNA in PBMCs and plasma by RT-PCR were monitored every week. The viral load in PBMCs ranged from 0.02 to 5.1 x 10^4 copies/ml (median 0.41 x 10^4).
Results: Clinical manifestations included fever, skin rash, thrombotic microangiopathy, seizures, bone and joint pain, hemophagocytosis, persistent thrombocytopenia, and several concurrent infections [bacterial (n=2): *Pseudomonas putida*, *Staphylococcus epidermidis*, *Rhizobium radiobacter*/fungal (n=2): *Aspergillus, Acremonium*/viral (n=4): CMV, EBV, BKV, RSV, *Rhinovirus*, *Boca virus*/parasitic (n=1): *toxoplasma gondii*. Despite the fact that the above manifestations could not always be attributed to HHV-6, it seemed that their emergence and/or aggravation correlated with its reactivation. Engraftment with ANC>microliter and PLTs>50,000/microliter occurred at median time of 19 (range 6-34) and 36 (range 41-95) days respectively. Two pts died before engraftment. Five pts received foscarnet and all but one responded at a median time of 10 days (range 9-19). The non-responder received cidofovir without response. In 3 pts, despite the reduction of viral load, viral DNA had been detected for several weeks. With a median follow up of 5 months (range 3-30), 3 pts died (systemic toxoplasmosis at d+52, leucoencephalopathy and sepsis at d+93, disease relapse at d+180).
Conclusions: A high incidence of HHV-6 reactivation with a variety of manifestations was noticed, that may increase immunosuppression and predispose to infections, but no clear conclusion could be made about its impact on engraftment. Further investigation is needed to identify risk factors for HHV-6 reactivation, and optimal prophylactic therapy.

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Fatal Disseminated *Bocavirus* Infection in a Young Transplant Patient

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Introduction: *Human bocavirus* (HBoV) is a recently discovered *parvovirus* frequently detected in respiratory samples from children during winter. Its role in immunocompromised patients remains unclear. We report a case of HBoV infection in a transplant recipient.
Materials, Methods and Results: A 33-year-old woman with acute lymphoblastic leukemia received an allogeneic hematopoietic stem cell transplant in March 2007, from an unrelated, 9/10 HLA alleles matched, donor. On day+306, while in ongoing remission, she presented with low grade fever, nasal congestion, neutropenia and thrombocytopenia. During this period she had been receiving tacrolimus, mycophenolate mofetil and methylprednisolone for persistent chronic GVHD. Anti-infective prophylaxis consisted of penicillin, posaconazole, acyclovir, atovaquone. Nasopharyngeal aspirates (NPA) were screened for 17 respiratory viruses (*influenza* A, B, C, *parainfluenza* 1, 2, 3, 4a, 4b, RSV A, B, *rinovirus*, *adenovirus*, *echovirus*, *bocavirus*, *coronovirus*, *metapneumonovirus* A, B) with PCR/DNA microarrays on day+306. HBoV was the only positive virus. Concomitantly, CMV reactivation by PCR was identified from blood. She was started on ganciclovir and one week later PCR for CMV was negative. The neutrophil count normalized within 10 days but thrombocytopenia and rhinitis persisted and the patient's general health was not improved. On d+ 339 her temperature rose to 39°C, dry cough and diarrhea ensued. d+ Chest radiograph and CT showed mild pericardial effusion. The bronchoalveoral lavage fluid was positive for HBoV without isolation of other pathogen. NPA remained HBoV positive. Intestinal tissue biopsy was not diagnostic, but PCR analysis of the intestinal tissue was positive for HBoV. On d+ 356 dyspnea and hypoxia were added. Chest radiograph showed diffuse infiltrations on both lungs. She died despite respiratory support.
Conclusion: HBoV caused a persistent, disseminated and finally fatal infection in that immunocompromised patient. Chronic GVHD and immunosuppressive therapy probably predisposed to viral dissemination. Further investigation is required for estimation of HBoV epidemiology and clinical manifestations in immunocompromised patients.

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Blood Stream Infections in Haematopoietic Stem Cell Transplant

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Background: Blood stream infections (BSI) remain an important complication of HSCT. The aim of this study was to analyze etiology, microbiological resistance and outcome of BSI after allogeneic HSCT.
Materials: Retrospective review of BSI in patients treated at HSCT Unit between 1/01/04 and 31/12/07.
Methods: BSI was defined as isolation of a pathogen from at least one blood culture. For common skin contaminants, 2 consecutive positive blood cultures were required. Survival after BSI was analyzed by univariate chi-square test.
Results: There were 169 episodes of BSI diagnosed in 132 patients (median 10 days after HSCT, range: -47; 4876) and 183 pathogens were isolated (Table 1).

Table 1. Etiology of 169 blood stream infections occurring between 01/01/04 and 31/12/07 in allogeneic stem cell transplant recipients

Organism	Number (%)
Gram-positive	**103 (56%)**
Staphylococcus	**45**
Coagulase negative	41
Staphylococcus aureus	4
Enterococcus	**40**
Enterococcus faecalis	21
Enterococcus faecium	17
Others (E. avium, E. species)	2
Viridans streptococci	**11**
Corynebacterium	**5**
Others (Streptococcus pneumoniae, Rothia mucillaginosa)	**2**
Gram-negative	**69 (38%)**
Escherichia coli	**25**
Pseudomonas aeruginosa	**20**
Klebsiella pneumoniae	**7**
Enterobacter	**7**
Stenotrophomonas maltophilia	**4**
Burkholderia cepacia	**3**
Others (2 Pseudomonas species, 1 Acinetobacter species)	**3**
Candida	**11 (6%)**
krusei	4
albicans	2
parapsilosis	2
species	2
glabrata	1